We claim:

1. A method of modulating inflammatory reactions involving leukocytes and leukocyte precursor cells in a subject comprising contacting a subject in need of said modulating with an amount of a xanthine oxidoreductase (XOR) inhibitor effective to modulate said inflammatory reaction mediated by leukocytes and leukocyte precursor cells in said subject.

2. The method of claim 1, wherein said leukocytes and leukocyte precursor cells are selected from the group consisting of mononuclear phagocytes, neutrophils and eosinophils.

3. The method of claim 1 wherein said inflammatory reaction is a disease selected from the group consisting of chronic heart failure, cardiomyopathy, diabetes, pancreatic inflammation, liver inflammation, Crohn's disease, uveitis, acute lung injury, COPD, sarcoidosis, granulomatous lung inflammation (GLI), acute lymphoblastic leukemia (ALL), ischemia reperfusion injury, hemorrhagic shock and renal transplant rejection.

4. The method of claim 3 wherein said inflammatory disease is granulomatous lung inflammation.

5. The method of claim 3 wherein said inflammatory disease is acute or chronic lung injury.

6. The method of claim 1 wherein said oxidoreductase inhibitor is selected from the group consisting of allopurinol, oxypurinol, tungsten, amflutizole, (-)BOF-4272 ([(-)-8-(3-methoxy-4-phenylsulfinylphenyl)pyrazolo(1,5-alpha)-1,3, 5-triazine-4-monohydrate]), diphenyleneiodonium dichloride, glutathione, glutathione precursors and dimethylthiourea (DMTU).

7. A method of modulating XOR activity in leukocytes and leukocyte precursor cells comprising contacting said leukocytes and leukocyte precursor cells with an agent which modulates the expression, synthesis, degradation, secretion, release, half-life, conversion

or catalysis of XOR in leukocytes and leukocytes precursor cells thereby modulating XOR activity.

8. The method of claim 7, wherein said leukocytes and leukocyte precursor cells are selected from the group consisting of mononuclear phagocytes, neutrophils and eosinophils.

9. The method of claim 7, wherein said contacting occurs *in vitro*.

10. The method of claim 7, wherein said contacting occurs *in vivo*.

11. The method of claim 7, wherein said leukocytes and leuckocyte precursor cells are involved in inflammatory reactions.

12. The method of claim 11, wherein said inflammatory reaction is an inflammatory disease selected from the group consisting of chronic heart failure, cardiomyopathy, diabetes, pancreatic inflammation, liver inflammation, Crohn's disease, uveitis, acute lung injury, COPD, sarcoidosis, granulomatous lung inflammation (GLI), acute lymphoblastic leukemia (ALL), ischemia reperfusion injury, hemorrhagic shock and renal transplant rejection.

13. The method of claim 12, wherein said inflammatory disease is granulomatous lung inflammation.

14. The method of claim 12, wherein said inflammatory disease is acute lung injury.

15. The method of claim 7, wherein said agent is selected from the group consisting of allopurinol, oxypurinol, tungsten, amflutizole, (-)BOF-4272, diphenyleneiodonium dichloride, glutathione, glutathione precursors and dimethylthiourea (DMTU).

16. A method of modulating inflammatory reactions involving leukocytes and leukocyte precursor cells in a subject comprising contacting said subject in need of said modulating with an amount of an agent which is effective to modulate the expression,

synthesis, degradation, secretion, release, half-life, conversion or catalysis of XOR in leukocytes and leukocyte precursor cells thereby modulating said inflammatory reactions.

17. The method of claim 16, wherein said leukocytes and leukocyte precursor cells are selected from the group consisting of mononuclear phagocytes, neutrophils and eosinophils.

18. The method of claim 16, wherein said inflammatory reaction is an inflammatory disease selected from the group consisting of chronic heart failure, cardiomyopathy, diabetes, pancreatic inflammation, liver inflammation, Crohn's disease, uveitis, acute lung injury, COPD, sarcoidosis, granulomatous lung inflammation (GLI), acute lymphoblastic leukemia (ALL), ischemia reperfusion injury, hemorrhagic shock and renal transplant rejection.

19. The method of claim 18, wherein said inflammatory disease is granulomatous lung inflammation.

20. The method of claim 18, wherein said inflammatory disease is acute lung injury.

21. The method of claim 16, wherein said agent is selected from the group consisting of allopurinol, oxypurinol, tungsten, amflutizole, (-)BOF-4272, diphenyleneiodonium dichloride, glutathione, glutathione precursors and dimethylthiourea (DMTU).

22. A method of modulating cytokine-induced inflammation in a subject comprising

a) removing leukocytes and leukocyte precursor cells from said subject to form a population of leukocytes and leukocyte precursor cells;

b) contacting said population of leukocytes and leukocyte precursor cells formed in part a) with an effective amount of one or more agents to obtain a treated cell population, wherein said one or more agents are effective to modulate the expression, synthesis, degradation, secretion, release, half-life, conversion or catalysis of XOR in leukocytes and leukocyte precursor cells; and,

c) administering said treated cell population of part b) to said subject;

wherein said administered cell population modulates said cytokine-induced inflammation in said subject.

23. The method of claim 22, wherein said leukocytes and leukocyte precursor cells are selected from the group consisting of mononuclear phagocytes, neutrophils and eosinophils.

24. The method of claim 22, wherein said agent is an inhibitor.

25. The method of claim 24, wherein said inhibitor is selected from the group consisting of allopurinol, oxypurinol, tungsten, amflutizole, (-)BOF-4272, diphenyleneiodonium dichloride, glutathione, glutathione precursors and dimethylthiourea (DMTU).